Characterization of quinolone resistance in *Salmonella enterica* serovar Indiana from chickens in China[1]

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ABSTRACT The aim of this study was to characterize the quinolone resistance of *Salmonella enterica* serovar Indiana isolated from chickens in China. A total of 293 *Salmonella* strains were isolated from chicken farms and slaughterhouses in Shandong province of China, and 130 (44.4%) were characterized as *Salmonella enterica* Indiana (chicken farms, n = 52 strains; slaughter houses, n = 78 strains). All isolate serotypes were tested with the Kauffmann–White classification system and examined for susceptibility to the quinolones: nalidixic acid, enrofloxacin, norfloxacin, and ciprofloxacin. The resistance of the *Salmonella* Indiana strains to nalidixic acid, enrofloxacin, norfloxacin, and ciprofloxacin were 100, 73.1, 71.2, and 82.7%, and 100, 59.0, 79.5, and 80.2%, respectively. Selected quinolone resistant strains were evaluated for mutations in genes (*gyr*A, *gyr*B, *par*C, and *mar*A) by DNA sequencing. The *gyr*A mutation was found in all isolates, the *par*C mutation was only found in some isolates, and the *gyr*B and *mar*A mutations were not observed. Quinolone resistance was evaluated in the representative isolates by screening for the quinolone resistance determinants, *qnr*A, *qnr*B, *qnr*S, *qep*A, and *aac* (*6′*)-*Ib-cr* using PCR technology. The quinolone resistance determinants in *Salmonella*, *qnr*A, *qnr*B, *qnr*S, and *qep*A were negative by PCR, but *aac*(*6′*)-*Ib-cr* had high detection rates of 90.4 and 96.2% in chicken farms and slaughterhouses, respectively. *Salmonella* Indiana containing the *gyr*A mutation was prevalent in farms and slaughterhouses and possessed a high frequency of the quinolone resistance determinant *aac*(*6′*)-*Ib-cr*. These bacteria may have originated from the same source.

Key words: *Salmonella* Indiana, quinolone resistance, *aac* (*6′*)-*Ib-cr*, *gyr*A

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INTRODUCTION

Salmonella is a serious zoonotic pathogen, which causes infectious diarrhea in humans and is the leading cause of bacterial foodborne outbreaks and incidences in China (Salam and Tothill, 2009). Most food poisoning events in China from 1994 to 2005 were caused by *Salmonella* (Wang et al., 2007). *Salmonella enterica* serovar Indiana is a highly pathogenic strain, which can cause acute enteritis, diarrhea, and death, as well as huge economic losses to the livestock and poultry industries (Yang et al., 2011). Quinolones are widely used due to their broad-spectrum antimicrobial activity and strong antibacterial effects in both human and animal treatment of diseases (Li and Li, 2002). Quinolone use has led to increasingly resistant bacteria (Hooper, 2001), and the isolation rate of multiple serotype *Salmonella* strains resistant to quinolones and fluoroquinolones has increased every year (Malorny et al., 1999). Taiwan, Japan, the Netherlands, and the United States have all experienced outbreaks of *Salmonella* resistant to quinolones (Helms et al., 2005). The resistance mechanisms of *Salmonella* to the quinolones have become increasingly complicated and have drawn worldwide attention (Michael et al., 2006). In recent years, new resistance mechanisms, including resistance to quinolones mediated by the quinolone resistance determinants of a pentapeptide repeat protein-encoding *qnr* (Strahilevitz et al., 2009), an efflux-pump-encoding *qep*A (Robicsek et al., 2006), and an aminoglycoside acetyltransferase-encoding enzyme variant *aac*(*6′*)-*Ib-cr* (Périchon et al., 2007) genes, have been found. These drug resistance genes are

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located on plasmids, which can allow the rapid spread of quinolone resistance to animals and humans. Horizontal gene transfer of plasmid-mediated quinolone resistance genes increases the threat to current treatments of infectious diseases.

The aim of this study was to characterize the quinolone resistance of *Salmonella* Indiana isolated from chicken farms and slaughter houses in different regions of the Shandong province in China.

MATERIALS AND METHODS

Salmonella Isolation, Identification, and Serotyping

A total of 1,024 samples were collected in 2009 from 2 chicken farms and 2 slaughterhouses in Shandong province of China. Fecal samples of 6-week-old healthy chickens from chicken farms were collected using sterile cloacal swabs, and the samples from slaughterhouse chicken meat were collected by washing the intact whole chicken carcasses with 100 mL sterile saline, followed by centrifugation at 5,000 × *g* for 10 min at room temp. The supernatants were then removed and the pellets were suspended with 100 μL sterile saline and incubated in selenite cysteine broth at 37°C for 24 h. From the total number of *Salmonella* strains (293, 28.6%) collected, 130 (44.4%) strains of *Salmonella enterica* serovar Indiana were isolated in a laboratory at the Institute of Animal Science and Veterinary Medicine, Shandong Academy of Agricultural Sciences. The strains were collected according to acquisition guidelines. The field studies did not involve endangered or protected species.

After the samples were incubated in sterile selenite cystine broth (Qingdao Hope Bio-Technology, Qingdao, China) at 37°C for 24 h, all incubated samples were grown on chromogenic medium for *Salmonella* (CHROM agar, CHROMagar Microbiology, Paris, France) at 37°C for 24 to 48 h. Only one colony/plate was picked; the purple-colored colonies on the culture plates were regarded as presumptive *Salmonella* colonies. Suspected colonies were isolated and grown on Difco nutrient agar (Becton, Dickinson, and Company, NJ), and then identified by transferring to tubes with triple sugar iron agar (Becton, Dickinson, and Company, NJ), lysin indole motility semisolid agar (Becton, Dickinson and Company, NJ), Voges Proskauer semisolid media (Becton, Dickinson, and Company, NJ), urease test broth (Becton, Dickinson, and Company, NJ), and Simmons citrate agar (Becton, Dickinson, and Company, NJ). At the same time, isolates were tested for the *inv*A gene by PCR methods (Wang et al., 2008).

Salmonella isolates were serotyped by slide agglutination for O and H antigens according to the Kauffmann–White scheme (Popoff and Le Minor, 1992) using antisera produced at the National Institute of Biological Sciences in Beijing.

Table 1. MIC interpretive criteria of the 4 quinolones.

		MIC (μg/mL)		
Quinolones	ATCC 25922	S (sensitive)	I (intermediary)	R (resistance)
Nalidixic acid	1 to 4	≤16	–	≥32
Ciprofloxacin	0.004 to 0.015	≤0.06	0.12 to 1	≥2
Enrofloxacin	0.008 to 0.03	≤0.25	0.5 to 1	≥2
Norfloxacin	0.03 to 0.12	≤4	8	≥16

Susceptibility Testing

The minimum inhibitory concentrations (**MICs**) of *Salmonella* Indiana isolates were determined by broth microdilution according to the Clinical and Laboratory Standards Institute guidelines (CLSI, 2007; CLSI, 2008). The *Salmonella* Indiana isolates were tested with the 4 antimicrobials, nalidixic acid (0.25 to 512 μg/mL), enrofloxacin (0.015 to 32 μg/mL), norfloxacin (0.03 to 64 μg/mL), and ciprofloxacin (0.015 to 32 μg/mL). The MIC interpretive criteria for the 4 quinolones are found in Table 1. *Escherichia coli* strain ATCC 25922 was used for quality control.

PCR Amplification and DNA Sequencing

PCR was used to analyze the 130 *Salmonella* Indiana isolates that also were quinolone resistant based on susceptibility tests. All primer sequences and the predicted sizes of the gene sequences are given in Table 2. The following antimicrobial resistance genes were evaluated in the quinolone resistant isolates: *qnr*A, *qnr*B, *qnr*S, *aac*(6′)-*Ib*-*cr*, and *qep*A genes were analyzed using previously described primers and PCR cycling conditions (Xia et al., 2010). The PCR analysis of each sample was carried out in a final volume of 20 μL containing 1 μL DNA template, 0.5 μL each primer at 10 nmol/L, 10 μL 2×PCR Master Mix (Tiangen Biotech, Beijing, China) and 8 μL double-distilled water. The variant *aac*(6′)-*Ib*-*cr* gene was further identified by digestion with the BstF5I restriction enzyme (New England Biolabs, Ipswich, MA). The obtained DNA sequences were compared with those in GenBank using Basic Local Alignment Search Tool (BLAST).

Mutations of Target Genes gyrA, gyrB, parC, and marA in Salmonella Indiana

Primers were designed according to the literature to amplify the *gyr*A, *gyr*B, *par*C, and *mar*A genes in the quinolone resistance-determining region (**QRDR**) of 6 of the 130 strains of *Salmonella* Indiana. Three *Salmonella* strains were selected from farms (Strains 433, L355, and 404) and slaughterhouses (Strains L68, 453, and 273) for evaluation of target gene mutations. The obtained PCR products of the selected strains were purified and sequenced. The online gene library comparison feature in GenBank was used to compare sequences and confirm that the previously mentioned DNA sequences matched the standard *Salmonella*

Table 2. PCR primers of resistance genes and the *gyr*A, *gyr*B, *par*C, and *mar*A genes in the QRDR.

Gene	Forward Primer (5′→3′)	Reverse Primer (5′→3′)	Annealing Temperature (°C)	Size (bp)
*qnr*A	TTCAGCAAGAGGATTTCTCA	GGCAGCACTATTACTCCCAA	57	500
*qnr*B	CCTGAGCGGCACTGAATTTAT	GTTTGCTGCTCGCCAGTCGA	58	671
*qnr*S	CAATCATACATATCGGCACC	TCAGGATAAACAACAATACCC	58.5	420
aac(6′)-Ib-cr	TTGCGATGCTCTATGAGTGGCTA	CTCGAATGCCTGGCGTGTTT	55	482
*qep*A	CCAGCTCGGCAACTTGATAC	ATGCTCGCCTTCCAGAAAA	54	500
*gyr*A	TATGCGATGTCGGTCATTGT	CACGAAATCCACCGTCTCTT	53.4	369
*gyr*B	GCGCTGTCCGAACTGTACCT	CGGTGATCAGCGTCGCCACTTCC	60	172
*par*C	TGCGTTGCCGTTTATTGG	TCGGCGTATTTGGACAGG	53.4	303
*mar*A	GCAACGCTTGAGTATTTGCT	CATTTTCATGGTGCTCTTCG	53.4	479

Typhimurium LT2 strain in the gene library. The corresponding DNA sequences of the tested bacteria were analyzed and compared to confirm mutation points and types.

RESULTS

Identification and Susceptibility Testing

The 130 strains collected from chicken fecal and meat samples were identified as *Salmonella enterica* serovar Indiana, of which 52 strains were from farms and 78 strains were from slaughterhouses. The remaining 163 isolates were identified as *Salmonella enterica* serovar Enteritidis. The only *Salmonella* serotypes identified were Indiana and Enteritidis in the 293 isolated strains.

The resistance rates of all quinolones tested in the *Salmonella* Indiana isolates from farms and slaughterhouses were 81.7 and 79.7%, respectively. The resistance rates of *Salmonella* from farms and slaughterhouses to the individual quinolones nalidixic acid, enrofloxacin, norfloxacin, and ciprofloxacin were 100, 73.1, 71.2, and 82.7%, and 100, 59.0, 79.5, and 80.2%, respectively. Analysis of the drug sensitivity results showed that the isolated *Salmonella* strains were generally resistant to several frequently used quinolones.

Detection of Genes Associated with Quinolone Resistance in Salmonella

PCR was conducted on templates of the 130 *Salmonella* Indiana strains to detect the quinolone resistance determininants *qnr*A, *qnr*B, *qnr*S, *aac(6′)-Ib-cr*, and *qep*A. The results of PCR analysis was negative for all resistance determinants except for *aac*(6′)*-Ib-cr*, which was as high as 90.4 and 96.2% in the farms and slaughterhouses, respectively (Table 3). The length of the fragment obtained by PCR amplification was 300 to 400 bp, which was consistent with the reported 346 bp for the *aac(6′)-Ib-cr* fragment (Lu et al., 2011). The PCR gene product was further identified by digestion with the BstF5I restriction enzyme. Finally, the sequence of the PCR amplification product was consistent with the *aac(6′)-Ib-cr* sequence as seen by comparison using the BLAST search on GenBank. This demonstrated that the target gene was *aac(6′)-Ib-cr*.

Table 3. Resistance genes in *Salmonella enterica* serovar Indiana from chicken fecal and meat samples by PCR.

Source	Strains	*aac(6′)-Ib-cr*	Positive Rate
Chicken farm	52	47	90.4%
Slaughterhouse	78	75	96.2%
Total	130	122	93.8%

Detection of Quinolone Target Gene Mutations

Primers were designed according to the literature to amplify the *gyr*A, *gyr*B, *par*C, and *mar*A genes in the QRDR of 6 *Salmonella* Indiana strains having different Pulsed Field Gel Electrophoresis (**PFGE**) patterns (Lu et al., 2011) isolated from farms (Strains 433, L355, and 404) and slaughterhouses (Strains L68, 453, and 273). All 6 strains carried the 4 genes (*gyr*A, *gyr*B, *par*C, and *mar*A), which is shown in Figures 1 and 2. The obtained PCR products were sequenced following purification and the mutations detected are shown in Table 4. Six different point mutations were detected in the 6 selected *Salmonella* Indiana strains, and *gyr*A and *par*C mutations were detected. The *gyr*A mutation was detected in all 6 strains tested, and was mainly in the loci of 83 and 87. The *par*C mutation was found in 5 of 6 *Salmonella* Indiana strains, but no strains had the *gyr*B or *mar*A mutations. These 6 strains were resistant to nalidixic acid, ciprofloxacin, enrofloxacin, and norfloxacin. The positive rate of the *gyr*A and *par*C point mutations in *Salmonella* resistant to the 4 quinolones was higher than that of the same gene point mutations in strains with other drug-resistant phenotypes, indicating that when there were more point mutations of helicase-encoding *gyr*A and topoisomerase-encoding *par*C in a *Salmonella* strain, the quinolone drug-resistance of the strain was higher.

DISCUSSION

Salmonella enterica serovar Indiana is pathogenic to animals and humans (Li et al., 2013). The 130 *Salmonella* Indiana strains isolated in the present study from chickens in Shandong province accounted for 44.4% of the total strains collected, which was about 16% higher than the 28.3% of the total strains collected in Hebi City, Henan province in 2010 (Zhang

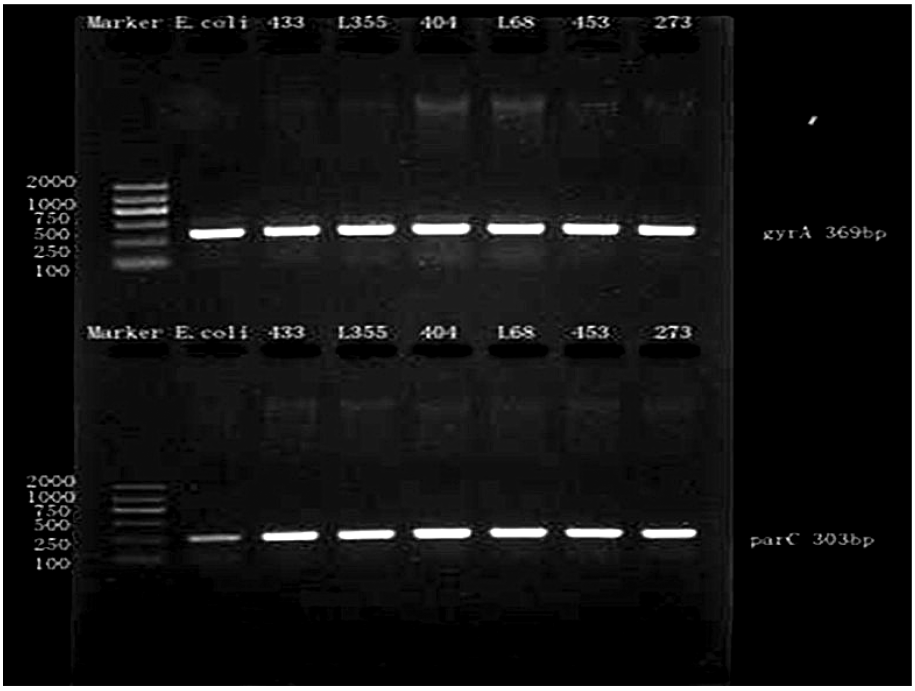

Figure 1. A gel photograph of the amplified PCR products from 6 *Salmonella* Indiana strains isolated from farms (Strains 433, L355, and 404) and slaughterhouses (Strains L68, 453, and 273) showing the presence of the *gyr*A and *par*C genes in the QRDR, and *E. coli* ATCC 25922 was used as control.

et al., 2010). These results demonstrate an increased prevalence of *Salmonella* Indiana in farms and slaughterhouses of Shandong province.

However, almost no information is available in the literature for *S.* Indiana. Recently, the emergence of multidrug resistant *S.* Typhimurium, *S.* Paratyphi, and *S.* Agona suggests that this multi-antibiotic-resistant phenotype may emerge in other *S. enterica* serotypes (Boyd et al., 2001; Meunier et al., 2002). Also, the few reports on *Salmonella* Indiana in the domestic and international literature have not described the existence of quinolone resistance genes. However, the *Salmonella* Indiana isolated in this study was not only widespread in farms and slaughterhouses, but also showed characteristics of quinolone resistance. Contaminated food from animal sources is a major cause of enteric *Salmonella* infections in humans (Gordon and Tucker, 1965; Thorns, 2000). During slaughter and processing, *Salmonella* from the gastrointestinal tract of carrier birds can contaminate other carcasses and the slaughter and processing lines (Rostagno et al., 2006). Although efforts have concentrated on controlling contamination within abattoirs, a high proportion of carcasses are still found contaminated with *Salmonella*. Recently, a study at University of California Davis demonstrated that 40% of the 120 participants involved with the preparation of raw poultry failed to cook their chicken to the required temperature of 74°C to kill *Salmonella* (Holland, 2014). This statistic demonstrates the importance of using proper food preparation methods when cooking poultry and the risks of quinolone resistance in *Salmonella* serotypes.

In an earlier study Wei and Chen (2005) isolated 68 *Salmonella* strains from animal and human sources in China, and examined them for susceptibility to 6 fluoroquinolones. They found the resistance rate of *Salmonella* Indiana to enrofloxacin and norfloxacin was 9.3 and 3.1%, respectively. In the present study, the resistance rate of *Salmonella* Indiana to enrofloxacin and norfloxacin was as high as 66.0 and 75.4%, respectively, which were much higher than previously reported. This indicates that *Salmonella* has produced stronger and more extensive drug resistance with the use of quinolones over time.

Park et al. (2006) found that *qnr*A, *qnr*B and *qnr*S coexisted in 313 strains of *Enterobacteria*, and 7/44 (15.9%) were *aac*(6′)-*Ib-cr* positive strains and 66/269 (24.5%) were *aac*(6′)-*Ib-cr* negative strains that possessed the *qnr* gene. In another study, among 101 strains from the family *Enterobacteriaceae* (comprised of 89 *E. coli*, 9 *Klebsiella pneumonia*, and 3 other genera) isolated from companion and food-producing animals, 34.7% of the strains were drug resistant to quinolones mediated by plasmids. The *aac*(6′)-*Ib-cr*, *qnr*, and *qep*A genes were detected alone or in combination in 19 (18.8%), 8 (7.9%), and 16 (15.8%) strains, respectively; and the genes *aac*(6′)-*Ib-cr* and *qep*A coexisted in 5 strains (Ma et al., 2009). Therefore, it can be assumed that these drug resistance genes may be present independently or jointly on the same plasmid, and play important roles in plasmid-mediated quinolone resistance.

Our study showed the *aac*(6′)-*Ib-cr* gene was detected in isolates from all sources. The *aac*(6′)-*Ib-cr* gene is a variant of *aac*(6′)-*Ib* and is responsi-

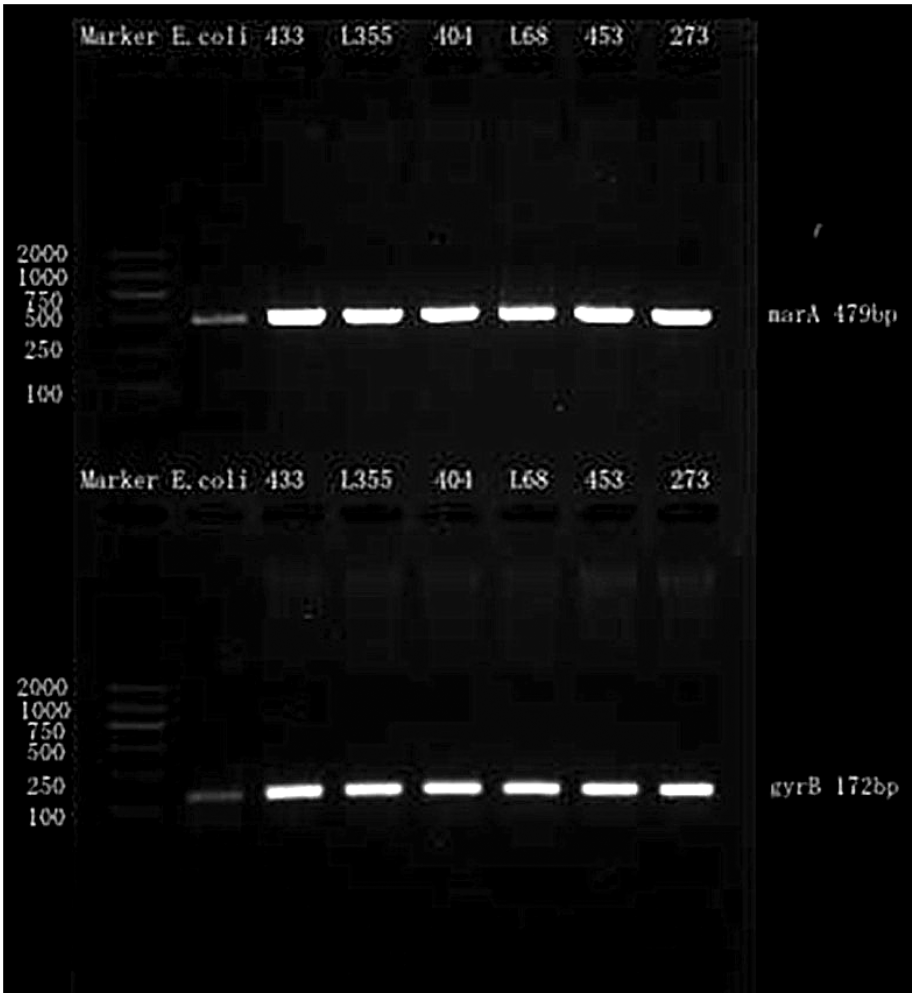

Figure 2. A gel photograph of the amplified PCR products from 6 *Salmonella* Indiana strains isolated from farms (Strains 433, L355, and 404) and slaughterhouses (Strains L68, 453, and 273) showing the presence of the *mar*A and *gyr*B genes in the QRDR, and *E. coli* ATCC 25922 was used as control.

Table 4. Mutation loci of amino acids from target genes and regulatory genes in *Salmonella enterica* serovar Indiana.[1]

				MIC (μg/mL)			
Source	Strain	Serotype	QRDR Mutation	NAL	CIP	NOR	ENRO
Chicken farm	433	Indiana	*gyr*A S83L *par*C S80I	>128	4	32	4
	L355	Indiana	*gyr*A S83I D87N	>128	2	32	2
	404	Indiana	*gyr*A T83I D87N *par*C S80I	>128	4	64	4
Slaughterhouse	L68	Indiana	*gyr*A T83I D87N *par*C S80I	>128	4	64	2
	453	Indiana	*gyr*A T83I *par*C S80I	>128	4	64	4
	273	Indiana	*gyr*A S83T *par*C S80I	>128	2	64	2

[1]QRDR = Quinolone resistance-determining region. Fluoroquinolone abbreviations are defined as the following: NAL = Nalidixic acid, CIP = Ciprofloxacin, NOR = Norfloxacin, and ENRO = Enrofloxacin.

ble for reduced susceptibility to ciprofloxacin and norfloxacin as a result of *N*-acetylation of the piperazinyl amine (Robicsek et al., 2006; Jiang et al., 2008). Ciprofloxacin is often used for treatment of salmonellosis. However, the incidence of fluoroquinolone resistance or reduced susceptibility in *Salmonella* species has increased worldwide over the past decade and there have been reports of failed treatments with ciprofloxacin (Aarestrup et al., 2003). The *aac*(*6′*)-*Ib-cr* gene also is widely distributed geographically

and is stable over time in the United States (Park et al., 2006). Our findings suggest that the *aac(6′)-Ib-cr* gene may have already been prevalent among bacteria before the emergence of the fluoroquinolone resistance *qnr* genes. By digestion with BstF5I and sequence analysis, the variant *aac(6′)-Ib-cr* gene was further identified. The *aac(6′)-Ib-cr* gene was highly prevalent in our *S.* Indiana isolates, and was present in 75 (96.2%) of the 78 isolates from slaughterhouses and 47 (90.4%) of the 52 isolates from chicken farms.

The variant *aac(6′)-Ib-cr* gene was reportedly the first enzyme conferring resistance to 2 structurally different antibiotic families (Courvalin, 2008), and the expression of *aac(6′)-Ib-cr* may facilitate the survival of DNA gyrase and topoisomerase IV mutants. In our study, the *gyr*A mutation was detected in all tested strains, and was mainly in the loci of 83 and 87. The *par*C mutation was found in 5 of 6 *Salmonella* Indiana strains, and no strains had the *gyr*B and *mar*A mutations. The point mutation positive rate for *gyr*A and *par*C in *Salmonella* resistant to all 4 quinolones was higher than that of the same gene point mutations in strains with other drug-resistant phenotypes, indicating that when there were more point mutations of helicase-encoding *gyr*A and topoisomerase-encoding *par*C in a *Salmonella* strain, the quinolone drug-resistance of the strain was higher.

In conclusion, a total of 293 *Salmonella* strains were isolated from chicken farms and slaughterhouses in Shandong province of China, and 130 (44.4%) were characterized as *Salmonella enterica* Indiana. The gene *aac(6′)-Ib-cr* had high detection rates of 90.4 and 96.2% in chicken farms and slaughterhouses, respectively. *Salmonella* Indiana containing the *gyr*A and *par*C mutation was prevalent in farms and slaughterhouses and possessed a high frequency of the quinolone resistance determinant *aac(6′)-Ib-cr*. The *gyr*B and *mar*A mutations were not observed. This indicated that the *Salmonella* Indiana isolated from these farms and slaughterhouses may have originated from the same source.

REFERENCES

Aarestrup, F. M., C. Wiuff, K. Mølbak, and E. J. Threlfall. 2003. Is it time to change fluoroquinolone breakpoints for *Salmonella* spp. Antimicrob. Agents Chemother. 47:827–829.

Boyd, D., G. A. Peters, A. Cloeckaert, K. S. Boumedine, E. Chaslus-Dancla, H. Imberechts, and M. R. Mulvey. 2001. Complete nucleotide sequence of a 43-kilobase genomic island associated with the multidrug resistance region of *Salmonella enterica* serovar Typhimurium DT104 and its identification in phage type DT120 and serovar Agona. J. Bacteriol. 183:5725–5732.

CLSI (Clinical and Laboratory Standards Institute). 2007. Performance Standards for Antimicrobial Susceptibility Testing, Seventeenth Informational Supplement. CLSI document M100-S17. Clinical and Laboratory Standards Institute, Wayne, PA.

CLSI (Clinical and Laboratory Standards Institute). 2008. Performance Standards for Antimicrobial Disk and Dilution Susceptibility Tests for Bacteria Isolated From Animals, Approved Standard, 3rd Ed. Informational Supplement. CLSI document M31-A3. Clinical and Laboratory Standards Institute, Wayne, PA.

Courvalin, P. 2008. New plasmid-mediated resistances to antimicrobial agents. Arch. Microbiol. 189:289–291.

Gordon, R. F., and J. F. Tucker. 1965. The epizootiology of *Salmonella menston* infection of fowls and the effect of feeding poultry food artificially infected with *Salmonella*. Br. Poult. Sci. 6:251–264.

DT104 Study GroupHelms, M., S. Ethelberg, and K. Mølbak. 2005. International *Salmonella* Typhimurium DT104 infections, 1992–2001. Emerg. Infect Dis. 11:859–867.

Holland, J. 2014. Home cooks caught on tape handling chicken unsafely. http://www.modbee.com/2014/07/01/34201. Accessed Jul. 9, 2014.

Hooper, D. C. 2001. Emerging mechanisms of fluoroquinolone resistance. Emerg. Infect Dis. 7:337–341.

Jiang, Y., Z. Zhou, Y. Qian, Z. Wei, Y. Yu, S. Hu, and L. Li. 2008. Plasmid-mediated quinolone resistance determinants *qnr* and *aac(6′)-Ib-cr* in extended-spectrum *β*-lactamase-producing *Escherichia coli* and *Klebsiella pneumoniae* in China. J. Antimicrob. Chemother. 61:1003–1006.

Li, G.-s., Y.-y. Wang, Z.-y. Zhu, H.-x. Wang, D.-h. Liu, W. Qiu, Y. Zheng, F.-q. Zhang, and Q.-s. Fan. 2013. Identification and drug resistance of the first *Salmonella* Indiana in Yunnan military garrison. Chin. Trop. Med. 13:1051–1053.

Li, W., and Z. Li. 2002. Development and present situation of veterinary quinolones. Chin. J. Vet. Med. 36:37–39.

Lu, Y., C. M. Wu, G. J. Wu, H. Y. Zhao, T. He, X. Y. Cao, L. Dai, L. N. Xia, S. S. Qin, and J. Z. Shen. 2011. Prevalence of antimicrobial resistance among *Salmonella* isolates from chicken in China. Foodborne Pathog. Dis. 8:45–53.

Ma, J., Z. Zeng, Z. Chen, X. Xu, X. Wang, Y. Deng, D. Lü, L. Huang, Y. Zhang, J. Liu, and M. Wang. 2009. High prevalence of plasmid-mediated quinolone resistance determinants *qnr*, *aac(6′)-Ib-cr*, and *qepA* among ceftiofur-resistant *Enterobacteriaceae* isolates from companion and food-producing animals. Antimicrob. Agents Chemother. 53:519–524.

Malorny, B., A. Schroeter, and R. Helmuth. 1999. Incidence of quinolone resistance over the period 1986 to 1998 in veterinary *Salmonella* isolates from Germany. Antimicrob. Agents Chemother. 43:2278–2282.

Meunier, D., D. Boyd, M. R. Mulvey, S. Baucheron, C. Mammina, A. Nastasi, E. Chaslus-Dancla, and A. Cloeckaert. 2002. *Salmonella enterica* serotype Typhimurium DT 104 antibiotic resistance genomic island I in serotype Paratyphi B. Emerg. Infect Dis. 8:430–433.

Michael, G. B., P. Butaye, A. Cloeckaert, and S. Schwarz. 2006. Genes and mutations conferring antimicrobial resistance in *Salmonella*: An update. Microbes Infect. 8:1898–1914.

Park, C. H., A. Robicsek, G. A. Jacoby, D. Sahm, and D. C. Hooper. 2006. Prevalence in the United States of *aac(6′)-Ib-cr* encoding a ciprofloxacin-modifying enzyme. Antimicrob. Agents Chemother. 50:3953–3955.

Périchon, B., P. Courvalin, and M. Galimand. 2007. Transferable resistance to aminoglycosides by methylation of G1405 in 16S rRNA and to hydrophilic fluoroquinolones by QepA-mediated efflux in *Escherichia coli*. Antimicrob. Agents Chemother. 51:2464–2469.

Popoff, M. Y., and L. Le Minor. 1992. Antigenic Formulas of the Salmonella Serovars, 6th revision. WHO Collaborating Center for Reference and Research on *Salmonella*, Institut Pasteur, Paris, France.

Robicsek, A., J. Strahilevitz, G. A. Jacoby, M. Macielag, D. Abbanat, C. H. Park, K. Bush, and D. C. Hooper. 2006. Fluoroquinolone-modifying enzyme: A new adaptation of a common aminoglycoside acetyltransferase. Nat. Med. 12:83–88.

Rostagno, M. H., I. V. Wesley, D. W. Trampel, and H. S. Hurd. 2006. *Salmonella* prevalence in market-age turkeys on-farm and at slaughter. Poult. Sci. 85:1838–1842.

Salam, F., and I. E. Tothill. 2009. Detection of *Salmonella* Typhimurium using an electrochemical immunosens. Biosens. Bioelectron. 24:2630–2636.

Strahilevitz, J., G. A. Jacoby, D. C. Hooper, and A. Robicsek. 2009. Plasmid-mediated quinolone resistance: A multifaceted threat. Clin. Microbiol. Rev. 22:664–689.

Thorns, C. J. 2000. Bacterial food-borne zoonoses. Rev. Sci. Technol. 19:226–239.

Wang, M.-y., M.-h. Xu, H.-w. Pan, and Z.-g. Wang. 2008. Establishment and preliminary application of LAMP *inv*A gene assay for rapid detection of *Salmonella*. Chin. J. Health Lab. Technol. 18:1971–1973.

Wang, S., H. Duan, W. Zhang, and J. W. Li. 2007. Analysis of bacterial foodborne disease outbreaks in China between 1994 and 2005. FEMS Immunol. Med. Microbiol. 51:8–13.

Wei, X., and Z. Chen. 2005. Studies on the minimum inhibitory concentration of six kinds of fluoroquinolones to *Salmonella*. Chin. J. Vet. Med. 39:4–8.

Xia, L. N., L. Li, C. M. Wu, Y. Q. Liu, X. Q. Tao, L. Dai, Y. H. Qi, L. M. Lu, and J. Z. Shen. 2010. A survey of plasmid-mediated fluoroquinolone resistance genes from *Escherichia coli* isolates and their dissemination in Shandong, China. Foodborne Path. Dis. 7:207–215.

Yang, B.-w., J.-l. Shen, M.-l. Xi, X.-l. Zhang, S.-h. Cui, X. Wang, and J.-h. Meng. 2011. Antibacterial susceptibility and subtypes of *Salmonella* isolates from retail chicken in Xi'an in 2007–2008. J. Food Sci. 32:130–136.

Zhang, X. L., B. W. Yang, X. G. Liao, X. L. Guo, and H. X. Chui. 2010. Antimicrobial resistance of foodborne and poultry-borne *Salmonella* in Hebi City, Henan province. Chin. J. Zoonoses. 26:1134–1136.